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NEWS	8	DEC 15	MEDLINE update schedule for December 2004
NEWS	9	DEC 17	ELCOM reloaded; updating to resume; current-awareness alerts (SDIs) affected
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NEWS	13	DEC 17	THREE NEW FIELDS ADDED TO IFIPAT/IFIUDB/IFICDB
NEWS	14	DEC 30	EPFULL: New patent full text database to be available on STN
NEWS	15	DEC 30	CAPLUS - PATENT COVERAGE EXPANDED
NEWS	16	JAN 03	No connect-hour charges in EPFULL during January and February 2005
NEWS	17	JAN 26	CA/CAPLUS - Expanded patent coverage to include the Russian Agency for Patents and Trademarks (ROSPATENT)

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                                                           ENTRY    SESSION
FULL ESTIMATED COST                                         0.42       0.42

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=> s stem cell and methylat?
L1          747 STEM CELL AND METHYLAT?

=> s l1 and py<2001
L2          383 L1 AND PY<2001

=> s l2 and (rlgs or bisulfite)
L3            9 L2 AND (RLGS OR BISULFITE)

=> duplicate remove l3
DUPLICATE PREFERENCE IS 'MEDLINE, BIOSIS'
KEEP DUPLICATES FROM MORE THAN ONE FILE?  Y/(N):n
PROCESSING COMPLETED FOR L3
L4            7 DUPLICATE REMOVE L3 (2 DUPLICATES REMOVED)

=> d 1-7 bib ab

L4   ANSWER 1 OF 7     MEDLINE on STN
AN   2001303754      MEDLINE
DN   PubMed ID: 11106238
TI   E-cadherin expression is silenced by 5' CpG island methylation
     in acute leukemia.
AU   Corn P G; Smith B D; Ruckdeschel E S; Douglas D; Baylin S B; Herman J G
CS   The Johns Hopkins Oncology Center, Baltimore, Maryland 21231, USA.
NC   CA-43318 (NCI)
     CA06973 (NCI)
SO   Clinical cancer research : an official journal of the American Association
     for Cancer Research, (2000 Nov) 6 (11) 4243-8.
     Journal code: 9502500. ISSN: 1078-0432.
CY   United States
DT   Journal; Article; (JOURNAL ARTICLE)
LA   English
FS   Priority Journals
EM   200105
ED   Entered STN: 20010604
     Last Updated on STN: 20010604
     Entered Medline: 20010531
AB   E-Cadherin is a transmembrane glycoprotein that mediates Ca2+-dependent
     intercellular adhesion in normal epithelium. In tumors of epithelial
     origin, E-cadherin expression frequently is reduced, an event that
     contributes to tumor invasion and metastasis. The role of E-cadherin in
     hematopoietic tissues is less clear. In normal bone marrow, E-cadherin is
     expressed on erythroid progenitors, CD34+ stem cells,
     and stromal cells, where it likely contributes to intercellular
     interactions during hematopoiesis. In this study, we used a nested-PCR
     approach to examine the methylation status of the E-cadherin 5'
     CpG island in blood and bone marrow samples from normal donors and in bone
     marrow from patients with acute leukemia. In normal peripheral blood
     mononuclear cells and bone marrow, E-cadherin was completely unmethylated.
     In peripheral blood mononuclear cells, expression was evident by reverse
     transcription-PCR. Immunoblotting confirmed E-cadherin protein expression
     in two lymphoblastoid cell lines derived from normal donors. In contrast,
     E-cadherin was aberrantly methylated in 4 of 4 (100%) leukemia
     cell lines, 14 of 44 (32%) acute myelogenous leukemias, and 18 of 33 (53%)
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acute lymphoblastic leukemias. Genomic **bisulfite** sequencing of primary leukemias confirmed dense **methylation** across the CpG island. **Methylation** was associated with loss of E-cadherin RNA and protein in leukemia cell lines and primary leukemias. Following treatment with 5-aza-2'-deoxycytidine, a **methylated** leukemia cell line expressed both E-cadherin transcript and protein. Our results show that **methylation** of E-cadherin occurs commonly in acute leukemia and suggests a hypothesis for E-cadherin down-regulation in leukemogenesis.

L4 ANSWER 2 OF 7 MEDLINE on STN DUPLICATE 1
AN 2000062851 MEDLINE
DN PubMed ID: 10593928
TI Tandem B1 elements located in a mouse **methylation** center provide a target for de novo DNA **methylation**.
AU Yates P A; Burman R W; Mummaneni P; Krussel S; Turker M S
CS Center for Research on Occupational and Environmental Toxicology, Oregon Health Sciences University, Portland, Oregon 97201, USA.
NC T32
SO Journal of biological chemistry, **(1999 Dec 17)** 274 (51) 36357-61.
Journal code: 2985121R. ISSN: 0021-9258.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 200001
ED Entered STN: 20000204
Last Updated on STN: 20000204
Entered Medline: 20000127
AB A cis-acting **methylation** center that signals de novo DNA **methylation** is located upstream of the mouse Aprt gene. In the current study, two approaches were taken to determine if tandem B1 repetitive elements found at the 3' end of the **methylation** center contribute to the **methylation** signal. First, **bisulfite** genomic sequencing demonstrated that CpG sites within the B1 elements were **methylated** at relative levels of 43% in embryonal **stem cells** deficient for the maintenance DNA methyltransferase when compared with wild type embryonal **stem cells**. Second, the ability of the B1 elements to signal de novo **methylation** upon stable transfection into mouse embryonal carcinoma cells was examined. This approach demonstrated that the B1 elements were **methylated** de novo to a high level in the embryonal carcinoma cells and that the B1 elements acted synergistically. The results from these experiments provide strong evidence that the tandem B1 repetitive elements provide a significant fraction of the **methylation** center signal. By extension, they also support the hypothesis that one role for DNA **methylation** in mammals is to protect the genome from expression and transposition of parasitic elements.

L4 ANSWER 3 OF 7 MEDLINE on STN
AN 1999428334 MEDLINE
DN PubMed ID: 10498621
TI Cloning and characterization of EphA3 (Hek) gene promoter: DNA **methylation** regulates expression in hematopoietic tumor cells.
AU Dottori M; Down M; Huttmann A; Fitzpatrick D R; Boyd A W
CS Queensland Institute of Medical Research, Department of Medicine, Herston, Queensland, Australia.
SO Blood, **(1999 Oct 1)** 94 (7) 2477-86.
Journal code: 7603509. ISSN: 0006-4971.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English

FS Abridged Index Medicus Journals; Priority Journals
EM 199911
ED Entered STN: 20000111
Last Updated on STN: 20000111
Entered Medline: 19991104
AB The Eph family of receptor tyrosine kinases (RTK) has restricted temporal and spatial expression patterns during development, and several members are also found to be upregulated in tumors. Very little is known of the promoter elements or regulatory factors required for expression of Eph RTK genes. In this report we describe the identification and characterization of the EphA3 gene promoter region. A region of 86 bp located at -348 bp to -262 bp upstream from the transcription start site was identified as the basal promoter. This region was shown to be active in both EphA3-expressing and -nonexpressing cell lines, contrasting with the widely different levels of EphA3 expression. We noted a region rich in CpG dinucleotides downstream of the basal promoter. Using Southern blot analyses with **methylation**-sensitive restriction enzymes and **bisulfite** sequencing of genomic DNA, sites of DNA **methylation** were identified in hematopoietic cell lines which correlated with their levels of EphA3 gene expression. We showed that EphA3 was not **methylated** in normal tissues but that a subset of clinical samples from leukemia patients showed extensive **methylation**, similar to that observed in cell lines. These results suggest that DNA **methylation** may be an important mechanism regulating EphA3 transcription in hematopoietic tumors.

L4 ANSWER 4 OF 7 MEDLINE on STN DUPLICATE 2
AN 1999044996 MEDLINE
DN PubMed ID: 9829531
TI High-resolution analysis of cytosine **methylation** in the 5long terminal repeat of retroviral vectors.
AU Wang L; Robbins P B; Carbonaro D A; Kohn D B
CS Childrens Hospital Los Angeles, Department of Pediatrics, University of Southern California School of Medicine, 90027, USA.
NC 1P01 CA59318 (NCI)
1R01 DK49000 (NIDDK)
SO Human gene therapy, **(1998 Nov 1)** 9 (16) 2321-30.
Journal code: 9008950. ISSN: 1043-0342.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199901
ED Entered STN: 19990128
Last Updated on STN: 19990128
Entered Medline: 19990114
AB Retroviral vectors based on the Moloney murine leukemia virus (Mo-MuLV) are among the most commonly used vectors for stable gene transfer into mammalian cells. However, expression from the transcription unit of the Mo-MuLV long terminal repeat (LTR) has often been unsatisfactory. Transcriptional suppression of retroviral vectors in vitro in embryonal carcinoma (EC) cells and in vivo in hematopoietic **stem cells** (HSCs) has been associated with increased levels of cytosine **methylation** in the vector 5' LTR. To obtain a comprehensive picture of the **methylation** pattern in the 5' LTR of retroviral vectors, we employed the **bisulfite** genomic sequencing technique, which allows detection of the **methylation** pattern of every CpG dinucleotide in a target sequence. We studied the 5' LTR within the Mo-MuLV-based vector, LN, and a series of multiply modified vectors, which show improved expression in vitro and in vivo. **Methylation** patterns of the vectors were compared in PA317 (3T3-derived) fibroblasts, which are permissive for expression from all of the vectors, and in F9 embryonal carcinoma (EC) cells, which are restrictive for expression from the parental Mo-MuLV LTR but show improved expression from the modified

vectors. These analyses revealed that the levels of **methylation** of CpG dinucleotides were globally consistent throughout the entire LTR, including the region of transcriptional factor binding. All vectors showed no measurable **methylation** of CpG dinucleotides throughout the 5' LTR in the PA317 fibroblasts. The CpG dinucleotides of the standard Mo-MuLV-based vector (LN) were highly **methylated** in F9 EC cells (49.1%). The doubly modified vector, MD-neo, which did not show improved expression, exhibited a relatively high level of **methylation** (45%), similar to that found in the LN vector. In contrast, the CpG dinucleotides of the triply modified vectors, which showed improved expression in EC cells (MND-neo and MTD-neo), were much less **methylated** (26.2 and 23.4%, respectively). The results extend our previous findings of an inverse correlation between gene expression and **methylation** of cytosine residues of the LTR of retroviral vectors.

L4 ANSWER 5 OF 7 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation. on STN
AN 1998:271100 BIOSIS
DN PREV199800271100
TI Sequence-specific **methylation** of the mouse H19 gene in embryonic cells deficient in the Dnmt-1 gene.
AU Warnecke, Peter M.; Biniszkiewicz, Detlev; Jaenisch, Rudolf; Frommer, Marianne; Clark, Susan J.
CS CSIRO Div. Mol. Sci., Sydney Lab., P.O. Box 184, North Ryde, NSW 2113, Australia
SO Developmental Genetics, (1998) Vol. 22, No. 2, pp. 111-121. print. CODEN: DGNTDW. ISSN: 0192-253X.
DT Article
LA English
OS Genbank-U19619
ED Entered STN: 24 Jun 1998
Last Updated on STN: 24 Jun 1998
AB We have used Dnmt-1c/c ES cells that are homozygous for disruption of the DNA methyltransferase gene to address how de novo **methylation** is propagated and whether it is directed to specific sites in the early embryo. We examined the imprinted H19 gene and the specific-sequence region implicated as an "imprinting mark" to determine whether de novo **methylation** was occurring at a restricted set of sites. Since the "imprinting mark" was found to be **methylated** differentially at all stages of development, we reasoned that the sequence may still be a target for the de novo **methylation** activity found in the Dnmt-1c/c cells, even though the loss of maintenance methylase activity renders the H19 promoter active. We used **bisulfite** genomic sequencing to determine the **methylation** state of the imprinted region of the H19 gene and found a low level of DNA **methylation** at specific single CpG sites in the upstream region of the imprinted H19 sequence in the Dnmt-1c/c mutant ES cells. Moreover, these CpG sites appeared to be favoured targets for further de novo **methylation** of neighbouring CpG sites in rescued ES cells, which possess apparently normal maintenance activity. Our data provide further evidence for a separate **methylating** activity in ES cells and indicate that this activity displays sequence specificity.

L4 ANSWER 6 OF 7 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation. on STN
AN 1998:92874 BIOSIS
DN PREV199800092874
TI DNA **methylation** in mouse A-repeats in DNA methyltransferase-knockout ES cells and in normal cells determined by **bisulfite** genomic sequencing.
AU Woodcock, David M. [Reprint author]; Linsenmeyer, Martha E.; Warren, William D.
CS Peter MacCallum Cancer Inst., Locked Bag No. 1, A'Beckett St., Melbourne, Victoria 3000, Australia
SO Gene (Amsterdam), (Jan. 5, 1998) Vol. 206, No. 1, pp. 63-67. print.

CODEN: GENED6. ISSN: 0378-1119.
DT Article
LA English
ED Entered STN: 25 Feb 1998
Last Updated on STN: 25 Feb 1998
AB Mouse ES cells with a null mutation of the known DNA methyltransferase retain some residual DNA **methylation** and can **methylate** foreign sequences de novo. We have used **bisulfite** genomic sequencing to examine the sequence specificity and distributions of **methylation** of a hypermethylated CG island sequence, mouse A-repeats. There were 13 CG dinucleotides in the region examined, 12 of which were **methylated** to variable extents in all DNAs. We found that: (1) there is considerable residual DNA **methylation** in ES cells lacking the known DNA methyltransferase (29% of normal **methylation** in the complete knockout ES DNA); (2) this other activity **methylates** at exactly the same CG sites as the major methyltransferase; and (3) differences in the distribution of **methylated** sites between A-repeats in these DNAs are consistent with this other activity **methylating** in a random de novo fashion. Also, the lack of any **methylation** in non-CG sites argues that, in other studies where non-CG **methylation** sites have been found by **bisulfite** sequencing, detection of such sites of non-CG **methylation** is not an inherent artifact in this methodology.

L4 ANSWER 7 OF 7 MEDLINE on STN
AN 97295815 MEDLINE
DN PubMed ID: 9151387
TI CpG **methylation** patterns in the 5' part of the nonclassical HLA-G gene in peripheral blood CD34+ cells and CD2+ lymphocytes.
AU Onno M; Amiot L; Bertho N; Drenou B; Fauchet R
CS University Laboratory for Hematology and Biology of Blood cells, University of Rennes I, France.
SO Tissue antigens, **(1997 Apr)** 49 (4) 356-64.
Journal code: 0331072. ISSN: 0001-2815.
CY Denmark
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199707
ED Entered STN: 19970716
Last Updated on STN: 19970716
Entered Medline: 19970701
AB A dominant goal of research focused on the nonclassical human leukocyte antigen G (HLA-G) gene is to understand the molecular mechanism involved in its limited expression. In the present report, we examined DNA **methylation** as a potential regulatory mechanism of HLA-G transcription in two cell types of the adult lymphomyeloid lineage: CD2+ lymphocytes express several mRNA isoforms while transcripts are undetectable in CD34+ hematopoietic cells. The **methylation** status of 63 CpG sites in the promoter and in the 5' CpG island was established using **bisulfite**-treated genomic DNA sequencing. **Methylation** was first analyzed by the direct sequencing of **bisulfite**-treated and amplified products. The general patterns of CpG **methylation** in the 5' part of the gene were found to be similar for CD34+ cells and CD2+ lymphocytes: the distribution of **methylation** was not uniform across the 63 CpG sites. In the promoter region, both CpG dinucleotides were partially or fully **methylated** whereas in the CpG island, several CpG sites were totally demethylated. Unexpectedly, in HLA-G positive CD2+ lymphocytes, a great number of CpG dinucleotides displayed a higher frequency of **methylation** relative to that found in CD34+ cells. However, the sequence analysis of cloned products revealed that the molecules have different **methylation** patterns which suggests that the HLA-G

gene is differentially expressed in CD2+ cells. Our results suggest that **methylation** is not the sole mechanism that achieves the repression of HLA-G transcription in immature CD34+ cells.